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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 09/826,319
Filing Date: April 03, 2001
Appellant(s): LAHN ET AL.

Rita P. Sanzgiri
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 4/14/10 appealing from the Office action mailed 4/6/07.

(1) Real Party in Interest

The examiner has no comment on the statement, or lack of statement, identifying by name the real party in interest in the brief.

(2) Related Appeals and Interferences

The examiner is not aware of any related appeals, interferences, or judicial proceedings which will directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal.

(3) Status of Claims

The following is a list of claims that are rejected and pending in the application:

Claims 1,2,9-32,34-36 are rejected.

Claims 3-8 are withdrawn from consideration.

(4) Status of Amendments After Final

The examiner has no comment on the appellant's statement of the status of amendments after final rejection contained in the brief.

(5) Summary of Claimed Subject Matter

The examiner has no comment on the summary of claimed subject matter contained in the brief.

(6) Grounds of Rejection to be Reviewed on Appeal

The examiner has no comment on the appellant's statement of the grounds of rejection to be reviewed on appeal. Every ground of rejection set forth in the Office action from which the appeal is taken (as modified by any advisory actions) is being maintained by the examiner except for the grounds of rejection (if any) listed under the subheading "WITHDRAWN REJECTIONS." New grounds of rejection (if any) are provided under the subheading "NEW GROUNDS OF REJECTION."

(7) Claims Appendix

The examiner has no comment on the copy of the appealed claims contained in the Appendix to the appellant's brief.

(8) Evidence Relied Upon

5,871,734	Lobb et al.	2-1999
5,869,448	Arrhenius et al.	2-1999
5,958,410	Wigzell et al.	8-1999
2002/0037286	Krause et al.	3-2002

Schramm et al., "Proinflammatory Roles of T-Cell Receptor (TCR)gammadelta and TCRalphabeta Lymphocytes in a Murine Model of Asthma", American Journal of Respiratory Cell and Molecular Biology, vol 22, (Feb 2000), pp 218-225.

(9) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

A) Claims 1,2,9-32,34-36 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The specification does not provide adequate written description of the claimed invention. The legal standard for sufficiency of a patent's (or a specification's) written description is whether that description "reasonably conveys to the artisan that the inventor had possession at that time of the. . .claimed subject matter", Vas-Cath, Inc. V. Mahurkar, 19 U.S.P.Q.2d 1111 (Fed. Cir. 1991). In the instant case, the specification does not convey to the artisan that the applicant had possession at the time of invention of the claimed inventions.

The claims encompass use of antibodies which bind TCR or CD3 or CD4 or CD8 from any mammal. Thus the claims encompass use of antibodies which bind the aforementioned molecules from any of the thousands of known mammalian species. Whilst the murine and human counterparts of the aformentioned molecules derived from mouse or humans were known in the art, there are thousands of mammalian species

wherein said molecules have not been isolated or characterized at the amino acid sequence level and wherein the identity of said molecules is unpredictable.

The skilled artisan cannot envision the detailed structure of the encompassed antibodies and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and a reference to a potential method of isolating it. In the instant application, the peptide itself is required. See *Fiers v. Revel*, 25 USPQ 2d 1601 at 1606 (CAFC 1993) and *Amgen Inc. V. Chugai Pharmaceutical Co. Lts.*, 18 USPQ2d 1016.

In view of the aforementioned problems regarding description of the claimed invention, the specification does not provide an adequate written description of the invention claimed herein. See The Regents of the University of California v. Eli Lilly and Company, 43 USPQ2d 1398, 1404-7 (Fed. Cir. 1997). In University of California v. Eli Lilly and Co., 39 U.S.P.Q.2d 1225 (Fed. Cir. 1995) the inventors claimed a genus of DNA species encoding insulin in different vertebrates or mammals, but had only described a single species of cDNA which encoded rat insulin. The court held that only the nucleic acids species described in the specification (i.e. nucleic acids encoding rat

insulin) met the description requirement and that the inventors were not entitled to a claim encompassing a genus of nucleic acids encoding insulin from other vertebrates, mammals or humans, id. at 1240. The Federal Circuit has held that if an inventor is "unable to envision the detailed constitution of a gene so as to distinguish it from other materials. . .conception has not been achieved until reduction to practice has occurred", Amgen, Inc. v. Chugai Pharmaceutical Co, Ltd., 18 U.S.P.Q.2d 1016 (Fed. Cir. 1991). Attention is also directed to the decision of The Regents of the University of California v. Eli Lilly and Company (CAFC, July 1997) wherein is stated:

"The description requirement of the patent statute requires a description of an invention, not an indication of a result that one might achieve if one made that invention. See In re Wilder, 736 F.2d 1516, 222 USPQ 369, 372-373 (Fed. Cir. 1984) (affirming rejection because the specification does "little more than outlin[e] goals appellants hope the claimed invention achieves and the problems the invention will hopefully ameliorate."). Accordingly, naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material. Thus, as we have previously held, a cDNA is not defined or described by the mere name "cDNA," even if accompanied by the name of the protein that it encodes, but requires a kind of specificity usually achieved by means of the recitation of the sequence of nucleotides that make up the cDNA." See Fiers, 984 F.2d at 1171, 25 USPQ2d at 1606.

B). Claims 1,2,9-32,34-36 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lobb et al. (US Patent 5,871,734) as evidenced by Arrhenius et al. (US Patent 5,869,448) in view of Schramm et al., Wigzell et al. (US Patent 5,958,410) and Krause et al. (US Patent Application Publication 2002/0037286).

Lobb et al. teach use of antibody against VLA-4 to treat asthma (see abstract).

VLA-4 is a receptor on T cells (see Arrhenius et al., column 63, last paragraph). AHR occurs in asthma (see column 12, Example 2). Lobb et al. teach aerosol administration of antiVLA-4 antibody (see column 12, Example 2). Lobb et al. teach use of humanized antiVLA-4 antibody (see column 5, penultimate paragraph). Said antibody does not stimulate T cell activation (said antibodies inhibit VLA-4 function, see column 7, penultimate paragraph). Lobb et al. teach use of monovalent antibody (see column 7, third paragraph). Lobb et al. teach use of antibody dosages encompassed by those recited in claims 18 and 19 (see column 6, penultimate paragraph). Lobb et al. teach administration of said antibody in PBS via nebulized spray (see column 6, penultimate paragraph). Lobb et al. teach the method of claim 27 (see claim 17). Lobb et al. teach the method of claims 28,31,32 (see column 12, Example 2). *Lobb et al. teach that the effect seen can be achieved without detectable blood levels of antibody (see column 12, last paragraph) wherein the antibody would not therefore substantially effect peripheral immune function (eg. because it was not present in the blood)*. Lobb et al. teach use of said method in humans (see claim 16). Lobb et al. teach that their method resulted in a

70% decrease in inhibition of late phase response which would correlate with the improved FEV1 as per claim 34. Lobb et al. do not teach use of antiTCR αβ antibodies. Schramm et al. teach use of IV antiTCR αβ antibodies to treat asthma (see abstract). Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract). Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (AKA pulmonary aerosol) administration of antiTCR antibody(see column 13, second paragraph and column 12, penultimate paragraph). It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Lobb et al. teach aerosol administration of an antibody which binds T cells to treat asthma and Schramm et al. teach that a different antibody which binds T cells (antiTCR αβ) can be used to treat asthma. One of ordinary skill in the art would have been motivated to do the aforementioned because Lobb et al. teach that the anti T cell antibody can be administered in a variety of art known routes including aerosol. One of ordinary skill in the art would have also been motivated to do the aforementioned because Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (AKA pulmonary aerosol) administration of antiTCR antibody(see column 13, second paragraph and column 12, penultimate paragraph). A

neutralizing antibody would have been used in the claimed method because Schramm et al. teach that asthma symptoms are reduced in the absence of TCR$\alpha\beta$ T cells (see abstract). Regarding the particular dosages of formulation or dosage per weight, a routineer would initially test a wide variety of different dosages in order to have determined the smallest effective dose of the antibody used. A routineer would have administered said antibody in conjunction with art known treatments for asthma such as those disclosed in column 2, first paragraph of Lobb et al. The antibody would have been administered either before or during asthma symptoms.

(10) Response to Argument

A) Claims 1,2,9-32,34-36 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Regarding appellants comments the claims encompass use of antibodies which bind TCR or CD3 or CD4 or CD8 from any mammal. Thus the claims encompass use of antibodies which bind the aforementioned molecules from any of the thousands of known mammalian species. Whilst the murine and human counterparts of the aformentioned molecules derived from mouse or humans were known in the art, there

are thousands of mammalian species wherein said molecules have not been isolated or characterized at the amino acid sequence level and wherein the identity of said molecules is unpredictable. The skilled artisan cannot envision the detailed structure of the encompassed antibodies and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and a reference to a potential method of isolating it. In the instant application, the peptide itself is required. See *Fiers v. Revel*, 25 USPQ 2d 1601 at 1606 (CAFC 1993) and *Amgen Inc. V. Chugai Pharmaceutical Co. Lts.*, 18 USPQ2d 1016.

In view of the aforementioned problems regarding description of the claimed invention, the specification does not provide an adequate written description of the invention claimed herein. See The Regents of the University of California v. Eli Lilly and Company, 43 USPQ2d 1398, 1404-7 (Fed. Cir. 1997). In University of California v. Eli Lilly and Co., 39 U.S.P.Q.2d 1225 (Fed. Cir. 1995) the inventors claimed a genus of DNA species encoding insulin in different vertebrates or mammals, but had only described a single species of cDNA which encoded rat insulin. The court held that only the nucleic acids species described in the specification (i.e. nucleic acids encoding rat insulin) met the description requirement and that the inventors were not entitled to a claim encompassing a genus of nucleic acids encoding insulin from other vertebrates,

mammals or humans, id. at 1240. The Federal Circuit has held that if an inventor is "unable to envision the detailed constitution of a gene so as to distinguish it from other materials. . .conception has not been achieved until reduction to practice has occurred", Amgen, Inc. v. Chugai Pharmaceutical Co, Ltd., 18 U.S.P.Q.2d 1016 (Fed. Cir. 1991).

Regarding appellants comments about the antibodies of Table 1 of the specification, the instant rejection has indicated that antibodies against the murine and human versions of the receptors were already known in the art. The antibodies referred to in the Table bind human versions of said molecules. However, the claims encompass antibodies that bind any mammalian species wherein the identity of said molecules in the thousands of other species of mammal is not disclosed in any of the prior art disclosed in the specification. Similarly the specification, page 3 does not even disclose what species of mammals were used to produce the antibodies referred to. The antibodies disclosed on page 13 bind human or murine receptors. The claims encompass use of antibodies which bind the aforementioned molecules from any of the thousands of known mammalian species. Whilst the murine and human counterparts of the aformentioned molecules derived from mouse or humans were known in the art, there are thousands of mammalian species wherein said molecules have not been isolated or characterized at the amino acid sequence level and wherein the identity of said molecules is unpredictable. None of the passages of the specification referred to disclose the structure of the receptors bound by the recited antibodies such that any

clue as to the nature of said receptors in species other than human or mouse is revealed. Whilst the murine and human counterparts of the aformentioned molecules derived from mouse or humans were known in the art, there are thousands of mammalian species wherein said molecules have not been isolated or characterized at the amino acid sequence level and wherein the identity of said molecules is unknown and unpredictable as per The Regents of the University of California v. Eli Lilly and Company, 43 USPQ2d 1398, 1404-7 (Fed. Cir. 1997). It also noted that the written description guidelines apply to method claims and product claims. Regarding appellants comments, Capon v. Eshhar does not address the issue under consideration (aka claims drawn to use of antibodies which bind mammalian T cell receptors wherein the prior art discloses antibodies which bind human or murine T cell receptors but wherein the claims encompass use of antibdoies which bind mammalian species wherein said molecules have not been isolated or characterized at the amino acid sequence level and wherein the identity of said molecules is unknown and unpredictable. Regarding appellants comments that the antigens were known and fully characterized in mammalian species, there is no current evidence of record to support said contention. Regarding appellants comments, as per Noelle v. Lederman, the claims lack written description in view of the evidence of record which indicates that only human or murine versions of the antigens bound by the antibodies recited in the claims were known in the art.

B) Claims 1,2,9-32,34-36 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lobb et al. (US Patent 5,871,734) as evidenced by Arrhenius et al. (US Patent 5,869,448) in view of Schramm et al., Wigzell et al. (US Patent 5,958,410) and Krause et al. (US Patent Application Publication 2002/0037286).

Regarding appellants comments about claim 36 and claim 1, Lobb et al. teach that the therapeutic effect seen can be achieved without detectable blood levels of antibody (see column 12, last paragraph) wherein the aerosol administered antibody would therefore not substantially effect peripheral immune T cell responses (eg. because it was not present in the blood). Regarding appellants comments about unexpected results, Lobb et al. teach that the effect seen can be achieved *without detectable blood levels of antibody* (see column 12, last paragraph) wherein the aerosol administered antibody would therefore *not substantially effect peripheral immune T cell responses* (eg. because it was not present in the blood).

Regarding appellants comments, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Lobb et al. teach aerosol administration of an antibody which binds T cells to treat asthma and Schramm et al. teach that a different antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma. In addition, Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary

aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (A.K.A. pulmonary aerosol) administration of antiTCR antibodies(see column 13, second paragraph and column 12, penultimate paragraph). Regarding appellants comments about motivation, Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (A.K.A. pulmonary aerosol) administration (see column 13, second paragraph and column 12, penultimate paragraph). In addition, one of ordinary skill in the art would have been motivated to do the aforementioned because Lobb et al. teach that the anti T cell antibody can be administered in a variety of art known routes including aerosol.

Regarding appellants comments about Lobb et al., *Schramm et al. teach that an antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma*. Thus, the art recognized that an antiTCR $\alpha\beta$ could be used to treat asthma. Furthermore, Lobb et al. disclose:

"For instance, to the extent that the beneficial effects reported herein are due to the inhibition of leukocyte recruitment to VCAM-1 expressing endothelium..." (column 8, last paragraph).

Thus, Lobb et al. contemplate that their method involves inhibition of leukocytes including T cells. Lobb et al. teach use of antibody against VLA-4 to treat asthma (see

abstract). VLA-4 is a receptor on T cells (see Arrhenius et al., column 63, last paragraph). Thus, the antibody taught by Lobb et al. binds T cells. Furthermore, *Schramm et al. teach that an antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma*. Regarding appellants comments about LFA-3, *Schramm et al. teach that an antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma*. Thus, the prior art already recognized that an antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma.

Regarding appellants comments about Schramm et al., Schramm et al. teach use of IV antiTCR $\alpha\beta$ antibodies to treat asthma (see abstract). Schramm et al. teach in the abstract that: "Similar results were obtained with antiTCR gammadelta or antiTCRalphabeta monoclonal antibodies." wherein the similar results refer to modulation of the inflammatory response seen in the mouse model for asthma (also see page 220,second column, last paragraph). Schramm et al. disclose that their results indicate that acute allergic responses are dependent on intact TCR$\alpha\beta$ T cells. The animals have asthma, receive the antiTCR antibody and the asthma related responses are resolved. Thus, the asthma is treated. Airway hyperresponsiveness is a component of asthma. Regarding the transgenic mouse experiments, the aformentioned quote indicates that similar results are obtained with antiTCRalphabeta antibodies. Furthermore, the only actual data provided in the specification involves mouse models. Thus, it is unclear as to why the mouse data provided by Schramm et al. is any less

relevant than the mouse data provided by applicant. Furthermore, there is no teaching in Schramm et al. that a complete systemic depletion of an entire T cell subset from an animal is required in the antibody treated animals. Lobb et al. teach use of antibody dosages encompassed by those recited in claims 18 and 19 (see column 6, penultimate paragraph). Regarding the particular dosages of formulation or dosage per weight, a routineer would initially test a wide variety of different dosages in order to have determined the smallest effective dose of the antibody used.

Regarding the Wigzell et al. reference, said reference discloses use of cytotoxic antiTCR antibodies which deplete T cells (see column 14, lines 4-7). Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (A.K.A. pulmonary aerosol) administration (see column 13, second paragraph and column 12, penultimate paragraph). Regarding appellants comments about evidence of the effectiveness of pulmonary administration, applicant is reminded that all art is deemed enabled in the absence of evidence to the contrary. The MPEP section 2121 discloses:

PRIOR ART IS PRESUMED TO BE OPERABLE/ ENABLING

When the reference relied on expressly anticipates or makes obvious all of the elements of the claimed invention, the reference is presumed to be operable. Once such a reference is found, the burden is on applicant to provide facts rebutting the presumption of operability. In re Sasse, 629 F.2d 675, 207 USPQ 107 (CCPA 1980). See also MPEP § 716.07.

There is no evidence of record that the Wigzell et al. reference lacks enablement.

Furthermore, the Wigzell et al. reference is used in the instant rejection in the context of the Lobb et al. reference which already discloses pulmonary aerosol administration of an antibody that binds a receptor found on T cells. Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract). Krause et al. teach:

"When antibodies are used therapeutically, a preferred route of administration is by pulmonary aerosol.".

This statement is not limited to a particular antibody taught by Krause et al. In addition, as per above, there is no evidence of record that the Krause et al. reference is not enabled. Lobb et al. teach that AHR occurs in asthma (see column 12, Example 2). Lobb et al. teach aerosol administration of antiVLA-4 antibody (see column 12, Example 2). Lobb et al. teach use of humanized antiVLA-4 antibody (see column 5, penultimate paragraph). One of ordinary skill in the art would have been motivated to do the aforementioned because Lobb et al. teach that the anti T cell antibody can be administered in a variety of art known routes including aerosol. One of ordinary skill in the art would have also been motivated to do the aforementioned because Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (A.K.A.

pulmonary aerosol) administration of antiTCR antibody(see column 13, second paragraph and column 12, penultimate paragraph).

Regarding appellants comments about Fahy et al., the comments in page 9 of said reference indicate that the reason that their antibody was not effective was because *it was an antibody that bound a soluble antigen (IgE) present in large quantities in the vascular space* wherein said IgE acted as a "sink of IgE". Fahy et al. hypothesize that the antibody might have been more immunogenic via the aerosol route, but the successful results of Lobb et al. would tend to disagree with this hypothesis. The issue of noncompliant patients is not germane to the instant discussion. The hypothesis that aerosolized antibody was not delivered in sufficient quantity to the lower airways seems unlikely as a potential problem for the claimed invention because the successful results of Lobb et al. would tend to disagree with this hypothesis. Therefore, *the most likely explanation for the results found by Fahy et al. is that their antibody was not effective was because it was antibody that bound a soluble antigen (IgE) present in large quantities in the vascular space wherein said IgE acted as a "sink of IgE".* The antibody used in the claimed invention *does not bind a soluble antigen*. The antibody used in the claimed invention binds alphabeta TCR found on the surface of T cells. There is no evidence of record that soluble TCR is found in large quantities in the vascular space wherein said TCR acted as a "sink". Therefore, the results of Fahy et al. are not germane to the claimed invention. Furthermore, Lobb et al. teach aerosol

administration of an antibody which binds T cells to treat asthma. In addition, Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (AKA pulmonary aerosol) administration of antiTCR antibody. (see column 13, second paragraph and column 12, penultimate paragraph). Regarding reasonable expectation of success, Lobb et al. teach aerosol administration of an antibody which binds T cells to treat asthma and Schramm et al. teach that a different antibody which binds T cells (antiTCR $\alpha\beta$) can be used to treat asthma. Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (A.K.A. pulmonary aerosol) administration of antiTCR antibody(see column 13, second paragraph and column 12, penultimate paragraph). Schramm et al. has already demonstrated that antiTCR$\alpha\beta$ antibody can be used to treat asthma. Lobb et al. have already used pulmonary administration of antibodies which bind T cells to treat asthma.

Regarding appellants comments about low dosage, the only claims that recited a dosage are claims 18-23. Claim 16 recites a dose of antibody per formulation, but *does not recite how much antibody is actually administered.*

The dosages of claims 18 and 19 are taught by Lobb et al. (for example see column 6, penultimate paragraph wherein the dose can be as low as 0.05mg/kg (aka 50 μg/kg). Thus, appellants comments regarding dosage are irrelevant to claims other than 20-23. Regarding claim 19 said claim does not recite "less than 40 ug per kg body weight" as per asserted by applicant. Said claim recites "less than about 40 ug per kg body weight". Regarding Lobb et al. and intravenous administration of antibody, the claims, the claims are not drawn to intravenous administration. Lobb teach pulmonary administration of the anti T cell antibody and that the dose can be as low as 0.05mg/kg (aka 50 μg/kg) (see column 6, penultimate paragraph). Regarding appellants comments about the sheep experiments in Example 2, said experiments are merely examples. Lobb teach pulmonary administration of the anti T cell antibody and that the dose can be as low as 0.05mg/kg (aka 50 μg/kg) (see column 6, penultimate paragraph). Furthermore Lobb et al. teach:

Dosages will vary depending on the sensitivity of the asthma sufferer to particular allergens, the concentration of allergen on exposure and frequency/duration of exposure(s), the proposed mode of administration (e.g., injection or inhalation), the desired plasma level of an agent, e.g., an antibody, the effectiveness of a particular agent, e.g., a particular antibody or combination of antibodies, in suppressing airway responsiveness, the clearance rate or half-life of the composition, and other such factors familiar to physicians experienced in the treatment of allergic asthma.

Regarding appellants comments about doses used and "low potency of antibodies", Wigzell et al. teach intrapulmonary administration of antibody (column 13, second paragraph) and that doses as low as 100 ng (aka nanograms) of antibody can be used (see column 13, first complete paragraph). Thus potential use of low doses of antibody was already well known in the art. Furthermore Lobb et al. teach:

Dosages will vary depending on the sensitivity of the asthma sufferer to particular allergens, the concentration of allergen on exposure and frequency/duration of exposure(s), the proposed mode of administration (e.g., injection or inhalation), the desired plasma level of an agent, e.g., an antibody, the effectiveness of a particular agent, e.g., a particular antibody or combination of antibodies, in suppressing airway responsiveness, the clearance rate or half-life of the composition, and other such factors familiar to physicians experienced in the treatment of allergic asthma.

Regarding US Patent 6,156,463 said patent does not contradict the teachings of dosages as per Wigzell et al. or Lobb et al. It also confirms that pulmonary administration of antibodies for treatment of allergy was well known in the art (see claim 13). In addition, the claimed invention encompasses a method of treating humans, but there is no disclosure in the specification of evidence that the dosages used in claims 20-23 would have any effect in humans. Thus, to the extent that applicant is arguing unexpected results, the results disclosed in the specification are not commensurate with

the scope of the claimed invention. In addition, the MPEP section 716.02(d) [R-2] states:

Unexpected Results Commensurate in Scope With Claimed Invention

Whether the unexpected results are the result of unexpectedly improved results or a property not taught by the prior art, the "objective evidence of nonobviousness must be commensurate in scope with the claims which the evidence is offered to support.".

Regarding appellants comments about the cellular specificity of the antigen bound by the antiVLA-4 antibody, given that said antibody binds T cells and that the antibody used by Schramm et al. binds T cells (antiTCR ab) and can be used to treat asthma, it is reasonable to conclude that the method of Lobb et al. using aerosol administration could be practiced using the antibody used by Schramm et al. that binds T cells (antiTCR $\alpha\beta$). Regarding appellants comments about advantages of the claimed invention, Krause et al. teach that antibodies which inhibit T cell activation are preferably administered via pulmonary aerosol (see section [0118] and abstract) and Wigzell et al. teach that pathologic T cells found in the lungs can be treated via intrapulmonary (AKA pulmonary aerosol) administration of antiTCR antibodies(see column 13, second paragraph and column 12, penultimate paragraph). Regarding

appellants comments about gamma/delta T cells, said species is not the elected species and is not currently under examination. Regarding claim 36, Lobb et al. teach that the therapeutic effect seen can be achieved without detectable blood levels of antibody (see column 12, last paragraph) wherein the aerosol administered antibody would therefore not substantially effect peripheral immune T cell responses (eg. because it was not present in the blood).

(11) Related Proceeding(s) Appendix

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

/Ron Schwadron/

Primary Examiner, Art Unit 1644

Conferees:

/Ram R. Shukla/
Supervisory Patent Examiner, Art Unit 1644

/Jeffrey Stucker/
Supervisory Patent Examiner, Art Unit 1649